华西协合大学医学院医学系毕业论文

急性胰腺炎

◆邓显昭

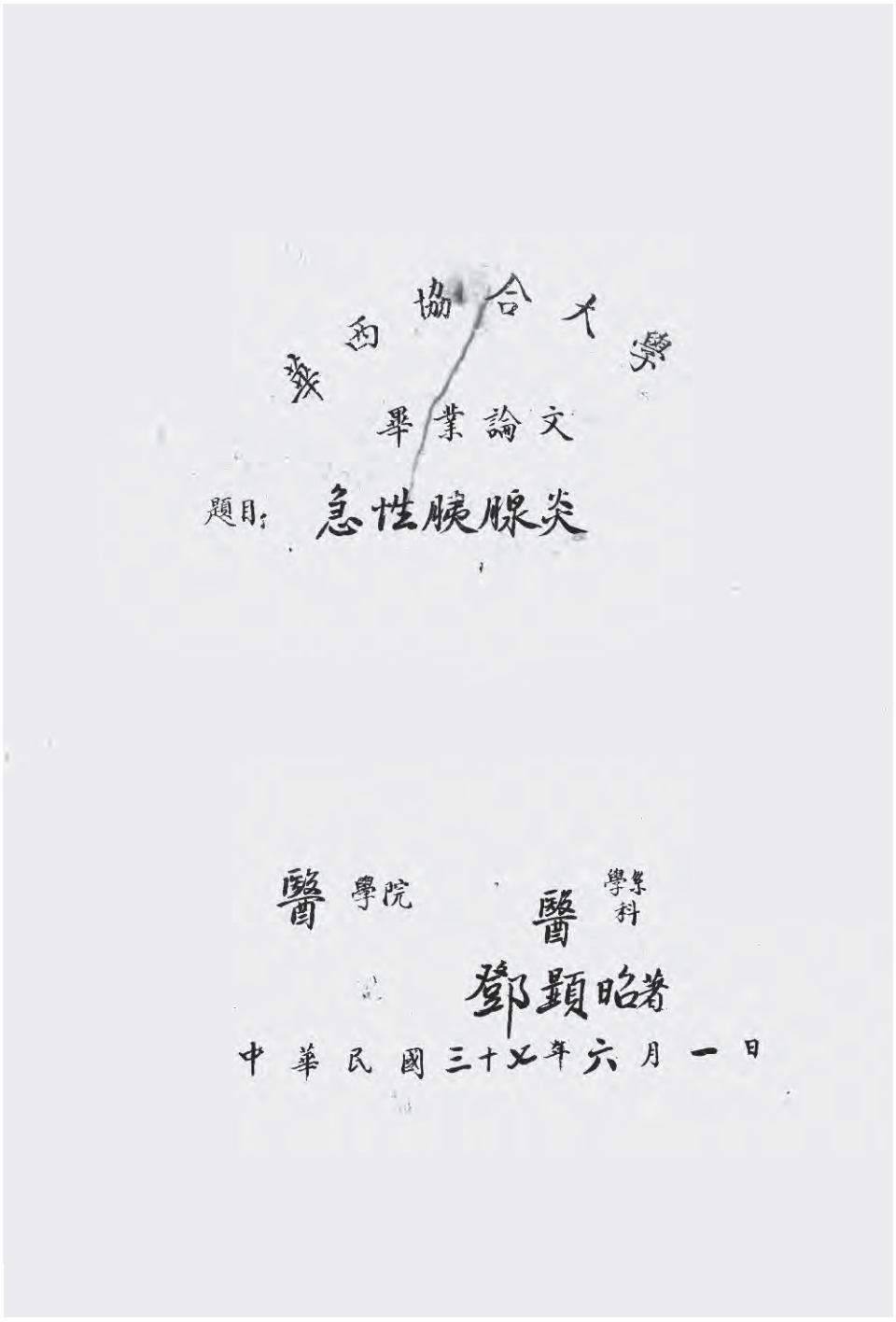
作者简介

邓显昭（1919—2014），原名邓建初，四川成都郫县唐昌镇（原崇宁县）人。1941年高中毕业，考入华西协合大学。1948年获得华西协合大学和纽约州立大学医学博士学位，在华西协合大学医院任住院医师。1951年随华西大学医疗队参加抗美援朝，同年受聘为外科学讲师。1954年建立泌尿专业组，任主治医师。1962年晋升为外科副教授。1972年任大外科副主任，后任四川医学院教务处教务长、图书馆馆长。1978年晋升为外科学教授，泌尿外科学研究生导师，是1978年后中国泌尿外科学第一批硕士、博士导师。

邓显昭一生和华西坝结缘，先后就读弟维小学、高琦中学、华西协合高中和华西协合大学医学院。他曾经回忆：“我们那届学了七年，从第一年学到第七年，真正一起毕业的从入学的90人中，只有10人毕业了。”在从医、执教生涯中，他将自己的毕生精力和智慧奉献给了祖国的医学教育事业和医疗卫生事业。邓显昭一生成绩颇丰，是我国泌尿外科界的知名专家，西南地区、四川省泌尿外科学的奠基人，华西医院泌尿外科的奠基人和杰出的学术带头人。1980年，任《中华泌尿外科杂志》《临床泌尿外科杂志》第一届编委。1981年4月，任成都市泌尿外科分会主任委员。1981年11月，任全国泌尿外科学会第一、二届常委。1986年11月，任四川省医学会泌尿外科分会第一届主任委员。后任成都市医学会泌尿专委会、四川省医学会泌尿专委会、西南地区泌尿协作组的名誉主任委员。他提出的“双瓣鱼嘴式”输尿管与直肠吻合的方法，有效减低了因吻合口狭窄或关闭不全导致梗阻、感染等并发症的发生率，其临床研

究成果达到国内领先水平。

2014年11月，邓显昭逝世，他将遗体捐献给了华西基础医学与法医学院解剖室，用作医学研究和教学之用。他生前治病救人，身后用超越死亡的博爱与奉献，指导着医学生去走医学的路。



華西協合大學

畢業論文

題目：急性胰腺炎

醫學院　　醫學科

鄧顯昭著

中華民國三十又三年六月一日

GRADUATION THESIS

PATHOGENESIS AND CLINICAL MANIFESTATION OF ACUTE PANCREATITIS WITH CASES ANALYSIS FROM JAN. 1945 TO AUG. 1947

BY

DEN HSIEN CHAO

IN

THE MEDICAL COLLEGE OF

WEST CHINA UNION UNIVERSITY

JUNE. 1948

ADVISOR: K. S. WANG M.D.
DEAN OF MEDICAL COLLEGE: C. L. TSAO M.D.
SUPERVISOR OF THE MEDICAL & DENTAL COLLEGE: L. G. KILBORN M.D.

CONTENT

INTRODUCTION

The first reference to the clinical entity, now known as acute pancreatitis, was made in 1882 when Balser described fat necrosis, Fitz in 1889 gave the comprehensive description of acute hemorrhaged pancreatitis. Popper In 1901 Opie demonstrated that injections of bile into the pancreatic duct of an animal would cause necrosis in the organ. In 1906 Flexner showed that the constituent of bile responsible for the necrosing effect in the pancreas was to be found in the bile salts. In 1913 Walters proved that spasm of the sphincter of Oddi would cause reflex of fluids introduced into the gall bladder of infected bile into the pancreatic duct.

Clinical support for the theory of bile diversion into the pancreatic duct is found in numerus statistics concerning the coincidence of pancreatitis with gall stone diseass. Reports of this nature have been published by Opie, Halsted, Körte, Ebner, Mayo Robson, Moynihan, Egdahl and many others; from these reports we may entertain the belief that on the one hand severe pancreatitis may occur either when the cystic duct is blocked, by stone or inflammation, and the pure liver bile is forced into the pancreas, or when the cystic duct having patent, the infected bile of the gall bladder, in cholecystitis, is expelled into the common duct and eventually into the pancreas. In cases of acute pancreatitis with apparently normal gall passage, the greater part of the necrosis of the pancreas in acute cases is believed to be due to self-digestion through activation of trypsinogen into trypsin. In 1899, Klippel, in 1908, Mauguerat and in 1914, Deaver were strongly believed that acute pancreatitis is caused by lymphatic transportation of infection from the gall-bladder, hemotogenous infection is also a probably cause of this disease.

It is generally recognized that the only proper treatment of acute pancreatitis is surgical, theoretically there are two aims should be considered, first of all will be the removal of the pathological lesion and preventing the absoption of split ppotein, the second one will be the prevention of recurrencer while the results of operation for acute pancreatitis vary ~~very~~ widely. It is not only that the type of operation performed differs from surgeon to surgeon; the type of case chosen for operations differs also, and the opinions of surgeons concerning the severity of [illegible] a given case are far from uniform, but in acute cases, according to the large reports given by Brocq, by Guleke and by Schmiden the mortality rate was 78 % before 1910, 68 % after 1910 (Broc[illegible] and from 1937--1945 the mortality reduced to [illegible].2 % (Sc[illegible]), 1924 52.2 % (Guleke). From the records

of the Royal Victoria Hospital in the years from: 1935--1944, 25 cases operated almost uniformly with a very limited drainage reaching to the surface of the pancreas and a cholecystectomy, the mortality was only 34 %. The figures given by those who insist strongly on the necessity of opening the swollen pancreas very thoroughly are no better.

Because the great disadvantage of wound disruption, formation of pancreatic fistula or complicated wound infections with much prolonged sickness, gradually a change to conservative treatment has occurred with an impressive reductions of the mortality rate and only a few experts still favor surgical treatment in the recent years. One can say that many more hopeless looking cases can be saved with proper conservative treatment than with surgery. However, conservative management can be applied safely only when all real surgical emergencies have been ruled out completely. (Popper)

Doubilet suggest to do endo-choledochal sphincterotomy for the cases of recurrent acute pancreatitis.

About 1938, Morton and his co-workers in Rochester, New York were struck by the interesting analogy between the parotid gland and the pancreas: both of these glands were of the serous type and blood amylase levels were elevated with disturbances of either. In 1940 Morton and Widget, encouraged by their early experiences with the roentgen treatment of acute pancreatitis, published their first ~~first~~ clinical report. In 1947, Chisholm reported from animal experiments a single small dose of 90 r is greatly helpful for the treatment of acute pancreatitis.

Totally 11 cases were carefully analysed ~~were~~ treated either conservatively or surgically, trying to make out a clear point upon the management of acute pancreatitis.

I am extremely grateful to my advisor Dr. K. S. Wang through his favorable help, the thesis is able to be completed.

EMBRYOLOGY

The pancreas is formed from neural endodermal evaginations. The first appear is the dorsal deverticulum which lies in close proximity to the hepatic diverticulum. In its grows, the dorsal diverticulum penetrates between the layers of mesogastrium forming the dorsal pancreas which afterwards becomes the body and the tail. The right and left ventral evaginations unite to form the ventral pancreas, grows to the left, in between the layers of mesogastrium to form the head of pancreas. At about the sixth week of the development, through the rotations of the duodenum around its long axis, the dorsal and ventral rudements approach one another and fuse together and forming a long slender glandular mass which passes backward within the mesoduodenum between the vertebral column and the greater curvature of the stomach. The dorsal pancreas is much larger than the ventral one and grows across the body toward the left until it reaches the spleen.

There are two stalkes in the embryonic pancreas. The stalk of the dorsal pancreas becomes the duct of Santorini, which empties into the posterior aspect of the duodenum less than one inch above the ampulla of Vater. It's terminal portion frequently atrophies and it remains the principal excretory duct of the pancreas in only 10 % of subjects (Moynihan and Mobson.). The stalk of the ventral pancreas becomes the duct of Wirrung the principal excretory duct which opens at the duodenal papilla either into the duodenum close beside the common bile duct or remains its embryonic relation, into the common bile duct itself. The originally more important duct of the dorsal pancreas drains only a portion of the head of the organ, and becomes the accessory pancreatic duct. In human adult it opens into the duodenum 1-3 cm. above the orifice of the common bile duct.

The primary diverticula give off buds which are lined with cylindrical epithelium and these inturn give off other buds and the process goes on until the gland is formed.

The islets of Langerhans are formed at a very early stage from the endodermal linning cells of some of the branching diverticula, which lose their connexion with the system of ducts.

The pancreas continues to be an intraperitoneal structure until the fifth month of intra-uterine life when its anterior dorsal covering disappears, making it retroperitoneal in position.

HISTOLOGY

Microscopic structure of the pancreas can be described briefly into two parts; the main one consisted branching tree of ducts, leading to acini of serous cells, the larger ducts, interlobular, are lined by a simple columnar epithelium with occasional goblet cells or pockets of mucous cells; the smallest ducts called intercalated ducts, consist of small, low cuboidal or flattened epithelial cells whose nuclei are oval and directed along the duct wall; the lumen is not much wider than the nuclei, but the acini are not always at the end of the ducts, those acini are raised from the repeatedly branched ducts, and they may bulge from the side of the duct. The duct cells have vesicular nuclei and clear non-granular cytoplasm. The acinar cells are typical serous cells, containning zymogen granules, mitochondria and Golgi apparatus. The nucleus is large, round and vesicular. In fact the pancreatic cell may be considered as the type of enzyme-secreting cells. In 1856 Claude Bernard first figured the shinny, refractive granules in the fresh pancreatic cells. The size and number of the granules vary with activity of the gland. In the resting condition they are large and closely packed in the inner end of the cell, down to the level of the nucleus. During digestion, the granules are discharged until only a few are present in the tip of the cells. The base of the cell is occupied by mitochondria and by chromidial substance which may cause the basal end of the cells to have a striated appearance due to avoidence of spaces occupied by poorly fixed mitochondria.

The smaller, but equally important part of the pancreas is the islet tissue first described by Langerhan in 1869, which scatter throughout the organ, differing from the acini in the arrangement and character of their cells. These islets of Langerhans occur in great numbers and vary in size from a single cell to groups of many hundreds. They are usually most in numerus in the tail of the pancreas and less frequent in its head.

Besides the ducts leading to the acini, there are present other smaller ducts often with obliteralted lumens which form anastomoses frequently with islets. From these islet cells new acini and new islet, probably develop.

The large islets are composed of irregular cords of cells, without lumen, separated from each other by large capillaries, which come into close contact with the bases of the cells. By special methods, the cells at least three types of cellular tissue can be differentiated, two of these contain five granules, the other is non-granular with smooth clear cytoplasm. The granules differ chemically; if the tissue is fixed in alchol, or in other watery fluids, another type of granule formalin, the granules of A (or alpha) cells are preserved; if fixed in other watery

fluids, another type of granule in the B (or beta) cells is present. The A cell is larger and polygonal, its nucleus unusally large and elliptical with little chromatin. B cell is much smaller, much more numerous and with a smaller spherical nucleus, centrally placed and with considerably chromatin. Some of the smaller islets are composed entirely of this type.

The relations of these cells to each other are not definitely known, but anyhow, the C cells (which are the cells have clear cytoplasma and no granules) may represent a younger form of the A cell. The B cells are much smaller, usually are completely filled with granules, and no relation between these granules and those of the A cells have been suggested.

Not only morphologically, the islets are distinct from the rest of the pancreas, physiologically also the two structures are quite different. The acini send their secretion through ducts to the intestine to aid in digestion; the islets are discrete ductless glands delevering their secretion, insulin, to the blood to regulate sugar metabolism. From the fact that the B granules are dissolved in alcohol, and that insulin is derived by alcoholic extraction, it may be inferred that the B cells of the islets are responsible for this active principle. The function of the A cells is not known.

As a whole, the pancreas somewhat resembles the parotid gland, it is devided into angular lobes and lobules by connective tissue septa, containing blood and lymphatic vessels, nerves and interlobular ducts. Along the ducts there is a thick coat of connective tissue with some muscle fibers and sometimes penetrate the ducts into the center of the lobules. Upon the blood supply of the pancreas, there are different opinions about the finer distributions. Some one said that acini and islets are supplied with different arterials and capillaries; but according to others , all the blood to the acini, must first pass through the islets, the presence of special arteriols to the acini being derived. The nerves ending around the blood vessels, ducts and pancreatic cells are chiefly non-medullated sympathetic fibers from the coeliac plexus, associated with scattered nerve cells found within the pancreas. The islets are said to be chiefly supplied by special nerves, not connected with those to the exocrine portion of the gland.

ANATOMY

The pancreas is a flattened and elongated organ, its weight range from 60 to 100 grams, measuring from 12 to 15 centimeter long. The broad right extremity is called the head and is connected with the body by a slightly constricted neck. The narrow left extremity forms the tail. It passes obliquely upwards and to the left, across the posterior wall of the abdomen. The head is lodged within the curve of the duodenum. At the angle of junction of the lower with the left lateral border there is a prolongation named the uncinate process, which project upward and to the left behined the superior mesenteric vessels. Anteriorly, the border between the head and neck, on right side, is a groove for the gastro-duodenal artery, on the left side, a deep notch where the superior mesenteric and splenic veins unite to form the portal vein. The posterior surface of the head of pancreas is in relation with the inferior vena cava, which runs upwards behind it and comes nearly the whole of this aspect. The uncinate process passes in front of the aorta. The neck of the pancreasse is only about 2 cm. in length. Its anterior surface is covered with peritonium and supports the pylorus. The gastro-duodenal and the superior pancreaticoduodenal arteries descend in front of the junction of the neck with the head. The posterior surface of the pancreas is in relation with the superior mesenteric vein and the beginning of the portal vein. The body of pancreas is somewhat prismoid in shape, and has three surfaces. The anterior surface is concave and is covered with peritonium and is separated from the stomach by the lesser sac. The posterior surface is devoidofrom peritoneum and is in contact with aorta and the origin of the superior mesenteric artery, the left suprarenal gland and the left kidney and its vessels. It is intimately related to the splenic vein, which courses from left to right and separate it from the structures mentioned. The inferior surface is covered with peritoneum; it lies upon the duodeno-jejunal flexure and on some coils of the jejunum. There is usually a process termed the tuber omentale projecting from the right end of the superior border. It is in relation above with the coeliac artery, from which the hepatic artery courses to the right just above the gland, while the splenic artery runs toward the left following a long course along this border. Along the anterior border, the two layers of the transverse mesocolon diverge from each other, one passing upwards over the anterior surface, the other backwards over the inferior surface. The superior mesenteric vesselsemerge under the right extremity of the inferior border which separates the posterior from the inferior surface. The tail is narrow and usually lies in between the two layers of the lienorenal ligment and in contact with the inferior part ofthe gastric surface of the spleen. It is also closely related to the splenic vessels.

The pancreatic duct traneverses the pancreas from left to right, lying nearer its posterior than its anterior surface. It begins by the junction of the small ducts of the lobules situated in the tail of the pancreas, and running from left to right through the body, receives the ducts of the various lobules composing the gland, when it reaches the neck of the pancreas, it turning downwards, backwards, and to the right, comes into relation with the bile duct, which lies to its right side. Together the two ducts pass obliquely into the wall of the second part of the duodenum, and there unite to form a short dilated duct, named the ampulla of Vater. The constricted distal end of this ampulla opens on the summit of the duodenal papilla, which is situated within this part of the duodenum at the junction of its medial and posterior wall, from 8 to 10 cm. distal to the pylorus. As a rule, the two ducts do not unite until they approach very close to the opening on the duodenal papilla. The pancreatic duct, near the duodenum, is about the size of an ordinary quill. Recent observations, mentioned by Elman cites the works of Meken who dissected 449 autopsy specimens and said a common channel between pancreatic and common ducts in the majority of cases (61 %). Thys making it possible for the reflex of bile into the pancreatic duct. Less than 40 %, the pancreatic duct and the bile duct open separately into the duodenum. Frequently, there is an additional duct which receives the ducts from the lower part of the head, and is known as the accessory pancreatic duct. It runs upward in front of the pancreatic duct to which it is connected by a communicating duct, and opens into the duodenum about 2 cm. above and slightly ventral to the duodenal papilla. The terminal part of the accessory duct may fail to expand and the secretion from the lower part of the head of the pancreas is then diverted along the communicating into the main pancreatic duct.

The arteries of the pancreas are derived from the splenic artery and from the pancreatico-duodenal branches of the hepatic and superior mesenteric arteries. The veins open into the portal, splenic and superior mesenteric veins. Its nerves, derived from the vagus and splanchnic nerves, reach it throught the splenic plexus.

The pancreas is drained by lymph vessels which enter the supra-pancreatic glands above the superior mesenteric root glands below, and the aortic glands behind.

Surgically, the head of the pancreas is at the level of the second lumbar vertebra, the neck is opposite the first lumbar vertebra, in the transpyloric plane and the body lies partly above that plane. The tail lies at the same level as the left flexure of the colon that is a little higher above the first lumbar vertebra.

—7—

Physiology

The pancreas has two signigicant functions: External excretion containing important digestive ferments and internal excretion concerned with c rbohydrate metabolism.

1. External excretion can be excited either by (a) Chemical stimulation or by (b) Nerve reflex, the former is the secretion juice which is more watery, alkaline in reaction and poor in enzymes, while the latter, vagus juice is viscid and richer in enzymes. The pure pancreatic juice is a colou/less transparent, viscous fluid which is alkaline (average Ph about 8) owing to the presence of $NaHCO_3$ (0.3-0.65%). 500-800 c.c. are secreted in 24 hours containing about 8 Gms. of Na Cl. The juice contains proteins (albumin and globulin) and three main enzymes:trypsin, lipase and amylase. The $NaHCO_3$ is present in such concentration that is meutralizes the acid present approximately equal volume of the gastric juice. The reaction of the intestine is thus prevented from becoming excessively acid. Trypsin is a powerful proteolytic ferment, acting best in an alkaline medium, which breaks down proteins to polypeptides and given fime, may liberate certain aminoacids, e.g. leucine, tyrosine and also curdles milk. Juice collected directly f om pancreatic duct without being allowed to come into contact with the duidunal mucosa is inactive, because it containes a precus or called trypsinogen. It is activated by a constituent of the sucus entericus called entero rinase which exerts a fe ment action on trypsinogen. Amylase of the pancreatic juice converts all forms of starch into maltose rapidly and completely In addition with the function of the succus entericus, all the dis cchorides will be converted into monosacharides, owing to the action upon the three chief ferments containing in th sucus entericus such are invertase, which converts the cane sug r into glucose and lactose converting lactose into glucose and galactose Lipase hydrolyses meutral fat to form one molecule of glycerine and and three molecules of fatty acid from each molecule of fat acted on. It is rapidly destroyed in vitro by trypsin. Protein had a protectige action, however, and this may account for the fact that lipase acts in the presence of trypsin in the intestine The activity of the enzyme is greatly increased by bile. The active constituents of bile are the bile acids which and hydrotro -pic substance that is they unite with fatty acids to form loose water soluble and diffusible chmmical compound which is stable in slightly acid solution and is unaffected by trypsin. The "complex" is also believed to diffuse equally freely into the lining cells of the intestinal villi, where it breaks up once more into it's constituent parts i.e. fatty acids and bile acids. It has to be assumed that the bile acids pass back to the intestinal surface of the villi to assets in the transfer of further quan-

titis of fatty acids. The pancreatic secretion is regulated first by the reflex stimulation of the vagi, in such stage, the pancreatic juice is increased for 10-20 minutes, within few minutes of the taking of food. Later on, the pancreatic juice is maintained at a high level by the stimulation of secretin which is probably normally present in the mucous membrane of the small intestine, under certain condition it is passed into blood stream, reached the pancreas via the circulation.

2. The internal secretion of the pancreas:- The Langerhams islets secret insaline which is a protein with a molecular weight of about 35000, it has been isolated in pure crystalline form. It destroyed by pepsin and trypsin, therefore it has always to be used parenterally.

PATHOLOGY

Grossly, in acute pancreatitis, the pancreasse is swollen soft, and dark in color. It may be red from hemorrhage or black from gangrenous. Hemorrhage is an accidental occurrence, which may or may not be present. Sometimes it dominates the picture. A pancreas may appear normal to the naked eye and yet may show the characteristic necrosis microscopically. The marked swelling is probably responsible for much of the severe pain. Small necrotic areas may be replaced by fibrous tissue. Large areas may be infected and form abscesses. The greater part of the pancreas may be destroyed. The peritonial cavity contains a characteristic fluid, dirty, fatty, and beef-juice appearced. The microscopical appearance is one of great necrosis of the acinar tissue, so that in the advanced cases no structure can be made out. A varying degree of hemorrhage takes places into thus necrotic tissue.

Fat necrosis is often seen in acute pancreatitis and is pathognomonic of that condition when encountered at operation. Small, dull, opaque, white areas are scattered over the surface of the pancreas and the surrounding omentum and mesentery. These represent areas of fat which have been broken down by the lipase in the liberated pancretic juice. Glyceral and fatty acid are formed, the glycerol is absorbed, and the fatty acids are deposited in the cells as acinar crystals. The areas tend to be absorbed in the course of few weeks, but the fatty acids may united with calcium, so that some of the patched may become calcified.

PATHOGENESIS

In review the history of acute pancreatitis, we should think about Fitz work, who first described the disease in 1889 at the Massachusetts General Hospital, as a clinical and pathological entity. He distinguished that disease into supurative, hemorrhagic and gangrenous form. The two later are rapidly fatal diseases, were shown to have a common pathology, gangrene following upon hemorrhagic necrosis if death were delayed even a few days. In both the hemorrhagic and gangrenous forms, fat necrowis almost always occurred both within the pancreatic capsule or into the peritoneal cavity. Suppurative pancreatitis, on the other hand, though similar in origin and onset, was found to run a longer and perhaps less fatal course. It rarely cause fatty necrosis.

Experimentally, Claude Bernard suggested giving injection of bile and sweetoil into the pancreatic duct to activate the secretion with in the pancreatic duct to produce self digestion. This hypophysis was confirmed from autopsy by Opie in case of biliary stone so placed as to encourage regurgitation of bile into the pancreatic duct. Archibald also supported Claude Bernard opinion from his experimental evidence that resistence at the papilla to an unusual and temporary rise of biliary pressu e may prevent the escape of bile into the intestine and force it into the duct of Wirsung, that is to say, only the spasm of the sphincter of Oddi might produce similar obstruction and reflex of bile for the explanation of cases inwhich no stone is found. Such as might arise reflexly in the same way as pylorospasm in the course of cholecystitis.

In the view of pathology acute pancreatitis is really pancreatic necrosis to which hemorrhage may or may not be added the real cause is still a matter of perennial dispute but anyhow the acute necrosis is due to the action of the pancreatic enzymes liberated from the ducts and usually is due to the passage of the infected bile along the pancreatic ducts, this activates the trypsinogen in the pancreas and converts it into trypsin. which proceeds to digest the pancreas more than one half the cases are associated with cholecystitis or calculi. Opie originally pointed out that if the common bile duct and pancreatic duct open into a common chamber (as occurs in 61% of persons of Mehnen's), impaction of calculus at the ampulls of Vatter will cause the bile to flow in to the pancreas. This is possible but very uncommon cause. The sphincter of Oddi as Archibald has shown that in the cat spasm of the muscle af the ampulla of Vatter is followed by a flow of bile into the pancreas, and such spasm may be produced by pressure on the gall bladder or by painting the ampulla with

weak cydrochloric acid. Mann has pointed out that in the human subject the sphincter is usually not placed distal to the entry of both ducts but proximal to the termination of the bile duct, and he suggests that here clinical imagination had preceded the demonstrated facts. Rich and Duff have suggested a different explanation. They found that in both human and experimental, hemorrhagic pancreatitis the constant and specific lesion was rapid nrcrosis of the walls of the arteries and veins, hemorrhage being due to rupture of the walls moreover they found that the pancreatic juice was able to produce necrosis without activation of the trypsinogen by intestinal content or bile, they believe that the machanism involved is rupture of dilated thinned out acini behind an obstructed ducts, this rupture being liable to occur from increased pressure in the ducts due marked secretion after a large meal. The main duct may be obstructed by a gall stone at the ampulla, but they found that the obstruction was usually in one of the smaller branches, being caused by metaplasia and piling of the lining epithelium. The lesion was present in over half the cases of hemorrhagic pancreatitis. The probable truth is that all of the factors out lined above may at times be responsible for producing the condition.

The cause of death is probably the poisonous split-protein products formed as the results of partial digestion of the pancreatic tissue. This products are absorbed very quichly, and this, together with the profound degree of shock perhaps acounts for the extraordinarily rapid termination of many of the cases.

Effects on the pancreas of stones in the common or cystic duct, stones in the common bile duct frequently affect the pancreas indirectly, dum partly to spread of infection from the wall of the ducts and partly because impacted stones in the common bile duct are capable of producing reflex of infected bile into the pancreatic ducts, is well known, the latter circumstance may lead to production of acute hemorrhagi-c pancreatitis. It is not uncommon to find subsiding condition of this sort, with fat necrosis in the vicinity, when exploration of the common duct is made for stones. Recurring edema of the head of panoreas is an even common surgical finding and there may be some demonstrable laboratory evidence of this fact in certain cases. Branch and Zollinger have noted the sharp rises in serum diastase which may follow immediately on biliary colic due to stone, these rises may occur even though no other sign of pancreatitis is present clinically(5).

Rienhoff and Pickrell reported recently that in 18% of their 250 dissections a complete block at the papilla would have converted the two ducts into a communicating system.

Leven in cholangiographic studies succeded to show filling of the pancreatic duct following injection into the common bile duct, thus proving common channel formation, in 23% of his cases On examination of 200 specimens of gall bladderbile of surgical cases without affection of the pancreas mostly cases of chole-lithiasis. The auther (H.L. Popper) found in 20 cases or 10% pancreatic enzyme in the gallbladder bile, thus proving that in 10% of these cases not only a common channel was present but that an actual reflux of pancreatic juice into the bile tract had occurred. In contrast to 10% incidence noted in the 200 cases with normal pancreas were the findings in 11 cases with present on post acute pancreatitis. In 16 of the 18 cases or in 89% common channel formation was demonstrated.

In 60-80% of cases of acute pancreatitis, associated pathology of the biliary tract is found which can account for the block of the papilla, while even in absence of any visible pathology of the biliary tract, as found in 20-40% of the cases of acute pancreatitis, a spasm of the sphincter of Oddi with formation of a common channel might be the causal mechanism of the acute pancreatitis. In 10-15% of the population, the possibility of common channel formation is present (12).

The intensity of the patholo ical process, the lack of histological evidence of inflammatory change in the fulminating cases, and the negative bacteriological findings, all point to the conclusion that the disease is not primarily due to bacteri-l infection. It is now generally accepted that the condition is essentially an acute necrosis of the pancreas and is due to self-digestion.

Trypsinogen can be activated by many agencies, including certain chemical substances and organisms such as B. coli, but infected bile is for the most potent substance.

These points would strongly proof that acute pancreatiti-s is not necessarily a real imflammatory change but some acute necrosis chiefly from reflux of bile into the pancreas (about 60% "10") or the so called self digestion of the pancreatic tissue and sometimes it may be caused by lymph-borne, blood-borne infections (as emboli), and sometimes may be caused by intrapancreatic obstruction due to hyperplasia of the pancreatic duct epithelium ("10") as Rich and Duff pointed out.

In the old teaching, it was described into hemorrhagic, gangrenous and purulent types,because such classification is somewhat misleading, in that gangrene or necrosis of the pancre-s is an invariable feature, whilst hemorrhage and suppuration are now regarded as secondary effects, therefore, in comformity with modern conceptions of the disease, it seems preferable to recognize two main variaties, the fulminating type, which is generally associated with hemorrhages, and the subacute type.

which may sometimes proceed to suppuration and generally there is no hrmorrhage.

From operation or autopsy of the fulminating cases, extensive necrosis of the pancreas with hemorrhage in the vicinity hemorrhagic or turbed effusion into the peritoneal cavity and fat necrosis spots over the omentum even more distant situations. Most of this cases, the whole pancreas is involve -d. Microscopically, the most conspicuous change isnecrosis of the parenchyma and interlocular septa with hemorrhages around the necrotic areas and in the surrounding arsolar tissue. Hemorrhages in some cases are so great as to force diffuse retroperitoneal hematoma. The hemorrhages are believed to occur as a result of degenerative changes brought about in the walls of blood vessels by emzymes liberated from the pancreas. Rich and Duff have shown attention to a peculier hyaline necro -sis of the vessel walls, affecting particularly the muscle fi -bers of the media and the interna l elastic lamina, and have shown that a similar lesion may be produced by the hypodermic injection of trypsin experimentally, The most characteristic change in acute pancretitis is fat necrosis occurring in the neigbourhood has been attributed to the action of lipase liberated from the pancreas and permeating along lymph vessels. The biliary tract often presents pathological changes in a-cute pancreatitis, stones are found in the gall bladder from 40-70% cases and it is remarkable that the stones are common -ly small in size. In occasional cases a stone is found in the common bile duct or impacted at the duodenal papilla.

Involvement of adjacent organs by the acute imflammatory process with in the gall bladder must also be considered Pancreatitis, when sufficient severity to give rise to symptom -s, is evidenced by a stuft of pain into the left upper quadrant of the abdomen and by an increase in the extension of pain to the lumbar region. Shock and extreme rigidity of the abdomen appear if actual hemorrhagic pancreatitis and fat necrosis develop ("6").

In the journal of S.G.O. Dec. 1947 Chisholni reported his work upon the experimental study of acute pancreatitis with special reference to X-ray therapy, using the same method described by Rick and Duff in 1936 in their classical study on the pathology of experimental pancreatitis in dogs, cannulated the duct of Santorini with blunt needle and bile, freshly aspirated from the gall bladder, was injected into the pancreas. When less than 2 c.c. of bile was slowly injected into 16 kilo -gram dogs at a pressure lower than 11 cm. of water, the ducta -l tree of the pancreas was filled but not ruptured, acute ede -ma of the pancrease developed few minutes later. The lobules became widely separated by a transparent transudate with which

microscopically showed a few extravasated leucocyte but primary an interacinar edema. When 2-10 c.c. of bile was as rapidly injected into dogs at a pressure over 18 cm. of water the duct -al tree of the pancreas was ruptured, the immediately vidible interlobular edema became bile tinged, few minates later small petechiae appeared in the acinar substance, microscpically large numbers of extravasated erythrocytes, leucocytes, fibrin and granular debris were found. Several hours later, numerous yellow plaques of fat necrosis were seen inside the pancreas as well as the surrounding tissue. By studying the serum amylase levels, Chisholm and his co-worker found that in dogs received 2-10 c.c. of bile under great pressure, the serum amylase level steadily rising while in dogs received less than 2 c.c. of bile under mild pressure, the serum amylase less acu -tely rised only. In the former blood amylase level continued to riseuntil the end of the 12 hours period of observation, while in the later, during the same interval, serum amylase was either approaching or had returned to normal levels.

CLINICAL MANIFESTATIONS OF ACUTE PANCREATITIS

Clinically, acute hemorrhagic and gangrenous pancreatitis usually manifested with severe epigastric pain accompanied by persistent vomiting and by shock of greater or less severity, depending upon the extent, a rapid thready pulse, a subnormal temperature which rises after the first few hours to perhaps 99 F., rarely high at any stage, cyanosis, almost unbearable distress following upon the pain, referred to the epigastrium and often to the back, and constipation as to suggest intestinal obstruction. Leucocytosis is only moderate. The abdomen contains bloody fluid and is everywhere very slightly tender, but most definite so over the pancrea itself. Especially if the tenderness, which is only present for a few days, is over the tail of this organ, does it distinguish pancreatitis from acute cholecystitis. Muscular resistance may obscure it. Serum amylase determination are of great importance in making a diagnosis. In the first days of the acute disease, moderate to large increases in amylase levels are characteristic. If the disease is not rapidly fatal these characteristic signs slowly subside merging into progressive emaciation and bodily deterioration, or local tenderness and swelling point to the formation of an abscess which either remains confined to the head of the gland, breaks into the lesser peritoneal cavity, or even ends by infiltrating the fatty tissue about the whole pancreas. (7"7")

The clinical picture of acute pancreatitis is not characteristic either in history , complaints, or in physical findings. Some features, howeverare prevalent and therefore, should be suggestive of pancreatitis. The disease is extremely rare in children but has been reported in all other age groups Some statistics show a greater incidence in man, some in women, in Popper's series, women were definitely predominent. Some cases are obese but a good many are not. Some are alcoholic, many are not. A history of previous gall bladder attacks or epigatric pain can be elicited in more than half of the cases. The patients are often subecteric, but as a rull have no distinct jaundice.

The most characteristic complaints are severe pain in the epigatrium radiating to the left side and to the back. This pain follows an episode of indigestion of a few days duration or many come suddenly, not so rarely after a heavy meal. One must not wait for the textbook discription of an excruciating pain not relieved by morphine to make the diagnosis of acute pancreatitis. The majority of cases have just very severe pain that is not easily distinguished from the other painful abdomen or renal conditions. However, the radiation to the left is diagnostic. There is often repeated vomiting without ensuring relief. The vomitus is uncharacteristic, containing food particles first, followed by gastric juice and bile, usually from the onset of the attack, the passage of gas and of stools is interrupted.

On physical examination, three types of cases can be found:

(1) Severe ,toxic cases which presents a shock picture . They have marked tenderness and some rigidity all over the abdomen, but more pronounced in the epigastric region and they have a high pulse rate and occasionally show mottled cyanosis of the abdomen or limbs. These cases look very much like acute vascular accients such as cormonary occlusion or embolism of the mesenteric vessels or they may appear like some overwhelming infection.

(2) Cases with predominantly peritoneal symptoms. The marked tenderness and marked rigidity will make differentiation from perforated ulcer difficult, but the rigidity rarely attains the board like quality of a perforation. This group comprises mild and severe cases and an early separation of the tow forms is often not possible.

(3) The last clinical form is characterised by intestinal paresis as the predominent symptom. These case show very little rigidity but some tenderness and a gradually increasing distention of the abdomen. They are usually light cases with rather limited constitutional symptoms. These cases look very much like renal or intestinal colics.

The laboratory findings are of greatest value for the diagnosis of acute pancreatitis. Urinalysis frequently shows albumin with various types of cylinders especially in severe cases. Glucosuria is seen in 5 to 20% of the severe cases, but rarely in mild cases. Trace of bilirubin are found occasionally in the urine and increased urinary urobilinogen is seen rather frequently. Blood examination often reveals a white blood count of around 20,000 with polymorphonuclears predominente. The N.P.N. and urea are at upper normal limits and are often markedly elevated in severe cases. There is frequently a slight elevation of icteric index. The fasting blood sugar is about normal in many cases. and as a rule markedly increased in severe forms. Glucose tolerance tests, though they regularly show abnormal ensues, are not advicable in acute cases. Edmondsen and Berue have recently reported a decrease of serum calcium in 36 to 60 cases of acute pancreatitis of which seemed to be in proportion to the amount of necrosis.

The most significant finding in acute pancreatitis is an increase of serum amylase and lipase, that can be found very shortly after the begining of the process. However the increase only lasts a few days then a gradual return to normal take place, regardless of the process in the pancreas. The increase amylase can be demonstrated in theurine too but variation in kidney function makes the blood test more reliable. Theincrease of serum amylase and lipase is due to absorption of pancreatic enzymes into the general circulation. The could show in anima l experiments that this absorption takes place mainly by way of portal vein and to a lesser extent through the thoracic duct.

and they could demonstrate that these absorbed enzymes are eliminated rapidly from the blood, that shows that the blood enzyme-level presents the actual balance of the continuously absorbed enzymes minus the continuously eliminated enzymes. Therefore, in actual pancreatitis, a constant inflow of large amount of enzymes must take place, to keep the blood level high despite the constant elimination. In a severely damaged pancreas, enzyme production decreases and finally stops, and therefore not enough enzymes are absorbed to maintain a high blood level. The explain the drop in blood enzyme levels after a few days, even though a progressive process may be taking place in the pancreas. Therefore it is important to do the enzyme test as soon as possible, for a negative test is of very little significance several days after the onset of the process.

The usual lipase determination takes 24 hours , the amylase test can be done in less the an hour and is therefore, of more practical value in acute cases. Increase of blood amylase is with few exceptions, limited to acute pancreatic processes. It is some times seen in gallstone attacks and occasionally, though only to a small degree, incases of peptic ulcers penetrating into the pancreas and in acute parotitis. Thus the serum amylase test, properly performed and evaluated will give us the means to make the quick diagnosis of acute pancreatitis in the majority of cases. It is an interesting but not too well known fact that amylase values are usually higher in the less severe cases and highest in edema of the pancreas, while only moderately elevated in pancreatic necrosis In contrast, the fasting blood sugar is not at all or only little above normal in mild cases and is increased in proportion to the extent of the process in severe cases. Agren and Lagerloff's secretion test, and the pancreatic function test elaborated in animal experiments are not suitable for cases of acute pancreatitis, for they are based on stimulation of the pancreatic secretion, which will aggravate the pathologic process in the pancreas. ("12")

According to Mc. Call, he considered as abnormal serum amylase values over 100 milligrams and less than 30 milligrems.

According to Walters and Clevelan, The values for diastase in the blood and in the urine usually are elevated after six or eight hours and remains so from seven to ten days. The most valuable test is that for the serum lipase, in 9[illegible] of a series of cases of acute pancreatitis, the value for lipase in the serum increased immediately after onset and over a period of three weeks gradually decreased to normal. ("15")

Ehrmann found experimentally, after the ligation of the pancreatic duct, the amylase in the urine and blood is highly increased for some time, but the quantity of diastase in the blood of the pancreatico-duodenal vein to be not higher than in other veins . Therefore Ehrmann assumed that diastase leaves the pancreas by way of lymph vessels.

Normally, insulin flows though the pancreatic duodenal veins into the general circulation. This mechanism can be proved by taken blood from such a vein from a dog, previously fed with carbonhydrates, and infused into another depancreatized dog to overcome the glucosuria for several hours. Therefore hyperglycemia and glucosura in acute disintergration of pancreas as in acute hemorrhagic pancreatitis is most likely due to the dextruction of the pancreatic tissue including the islets of Langerhams rather than obstruction of the pancreatico-duodenal duct.

In case of obstruction, the decrease or lack of pancreatic ferments can be determined by means of a duodenal tube and examination stumilation of pancreatic secretion by previous administration of secretion.

~~Obstruc~~ Observation of the feces ~~by/previous/adminstra-of/secretion//~~ after taking of some special kind of foods, which are digested exclusively by the pancreatic ferments. The deficiency of the pancreatic ferments may well be overlooked. It is also important to find out undigested fat particles and undigested meat fibers in the stool to confirm particle and undigested meat fibers in the stool to confirm the lack of steapsin and trypsin. ("14")

K.F.Herfort studied the case history and autopsy of 29 cases suffering from acute pancreatitis necrosis who were treated in the Hospital of the Charles University of Prague. He found that an absolute lymphocytopenia was present in all the cases and this lymphopenia is especially helpful in establishing the diagnosis when the W.B.C. is normal or the amylase level is not significantly changed ("13").

TREATMENT OF ACUTE PANCREATITIS

On discussing the treatment of acute pancreatitis, there are different opinions. According to Christopher, immediate operative treatment should be undertaken, if the patient is seen in the first 48 hours; after this time it may be wise to wait until a localized collection is formed. While Homans said that in case of acute pancreatitis, conservative treatment as infusion of plasma, and normal saline, large doses of sedatives, inlying of Miller-Abbott tube, is always indicated. In such cases if do not improve, operation in the form of biliary tract drainage may be helpful, as may drainage of an abscess or necrotic area, if such exist. He also said that incision of the pancreas itself is harmful.(7) (8)

Formerly, acute pancreatitis was considered as a surgical emergency, the mortality of such treatment including incision and drainage of the pancreas or drainage of the gall-bladder or common duct ranged around 50%. Gradually a change to conservative treatment has occurred with an impressive reduction of the mortality rate. An important disadvantage of surgery in severe cases is also the rather frequent incidence of wound disruption, formation of a pancreatic fistula or of complicated wound infection with much prolonged sickness. Conservative management can be applied safely only when the diagnosis correct beyond any doubt and when all real surgical emergencies have been ruled out completely. (12)

The proper conservative therapy has its aim to diminish the secretory activity of the pancreas and to relieve spasm of the Sphincter of Oddi. Anything that stimulates the vagus nerve will stimulate the pancreas; therefore, prostigmine as well as morphine or its derivatives, should be excluded. The vagotonic action of morphine might conceivably be the reason for the often mentioned insufficient pain relief after morphine administration in acute pancreatitis. Hyperglycemia has a pronounced stimulating effect by way of the cholinergic mechanisms and hypoglycemia has a marked vagotonic action too. Parenteral glucose administration as well as the regulation of the blood sugar by insulin must therefore be handled extremely carefully. In order to provoke secretin stimulation of the pancreas, the patient should not get anything by mouth. Continuous suction should keep the stomach empty and prevent hydrochloric acid from entering the duodenum. To inhibit pancreatic secretion, ephedrine and repeated doses of atropine are indicated. Intravenous fluids should consist mainly of saline solution and blood plasma. Glucose should be given only with repeated checks of the blood sugar and combined with atropine.

Pupper did try paravertebral block with novocaine on the left side at the level of the 8th. to 10th. thoracic spinal process. In 4 of 7 cases the block had not only a diagnostic but also a remarkable therapeutic effect.

For the purpose in present recurrences, it seems best to have the patient avoid heavy, rich meals, or any food that might irritate the bile tract.

In cases with biliary tract pathology, surgery is indicated, because this will eliminate a possible cause of further attacks. (12)

Because of the similarity of the pancreas to parotid in structure, it has been suggested by Morton and Widger (1938 to 1940) that inflammation of the pancreas will respond equally favorably to x-ray therapy. In 1945, there were 8 cases of acute pancreatitis treated by roentgen therapy in the Peter Bent Brigham Hospital in Boston together with the 13 cases then reported in the literature.

In 1947, Chishohn reported his experiment upon the x-ray treatment in acute pancreatitis by studying the response of normal pancreas to x-ray therapy as reflected by alternation of the blood amylase levels and found that an appropriately small single dose of Roentgen rays administered to dogs having experimentally induced pancreatic necrosis appears to inhibit amylase enzyme production with minimization of pancreatic tissue destruction rather than a large single dose or repeated serial exposures of x-ray to pancreas. From such results, the application of these observations to the Roentgen treatment of acute pancreatitis in humans is clearly suggested. (11)

In case of acute pancreatic edema may cause most severe and intractable abdominal pain, one can readily distinguish two components. The first is visceral, due to the direct or transmitted stimulation of the celiac ganglion, accompanied by girdle pain around the waist line and radiating fanwise to the lower abdomen. While, when the lesion extends to the peritonium, the somatic fibers of the lower intercostal or upper lumbar nerves or even the branches of the lumbo-sacral plexus are irritated. This somatic pain shows skin hyperesthesia in the appropriate segments and sensitivity of the spinous processes of the segmented zones. Muscle rigidity may appear. The visceral pain is continuous, but shows intermittent, lacerating exacerbations.

In that article, the author reported 2 cases in which unilateral splanchnic nerve section with lower dorsal sympathectomy was done for intractable pain. One of them was a case of a chronic recurrent calcareous pancreatitis with exacerbations, the operation resulted in sudden relief of pain, which could not be controlled with heavy doses of narcotics taken over a period of 15 months. So, it is emphasized in intractable upper abdominal pain of visceral origin, paravertebral block, followed if necessary by splanchnicectomy is valuable. (17)

Since 1901, when Opie focused the attention of surgeon on the observation that acute pancreatitis could be produced by retrojection of bile into the pancreatic duct, many surgeons did try cholecystectomy and choledochostomy in cases without stone in the biliary tract as well as the stone or stones definitely occurring. Because (1) the removal of a muscular organ would prevent the forceful propulsion of concentrated bile into the pancreatic duct, (2) the absence of concentrated gall bladder bile would diminish the severity of its necrotizing action, and (3) cholecystomy often resulted in relative atonicity of the sphincter of Oddi. These concepts were confirmed experimentally by Wangesteen on cats. Under his experimental condition, the incidence of acute pancreatitis was reduced 50 per cent by cholecystomy. But since the essential anatomy a common passage way between the choledochus and the pancreatic duct remains unchange in all the operative prodedures, it is obvious that the possibility of further obstruction due to factors other than stone might be anticipated. The recurrence of pancreatitis in patients after these operations confirms this fact.

One of the factors other than the presence of stone which might also cause obstruction of the biliary tract is the spasm of the sphincter of Oddi. From the recent cholangeographic studies on patients have confirmed that spasm of this muscle was occasionally found post-operatively and such spasm also could be produced by given drugs as morphine and local application of hydrochloric acid.

Obviously, if obstruction at the ampulla of Vater by spasm converts the bile and pancreatic ducts into a common channel leading to the production of acute pancreatitis, the answer would be to cut the sphincter of Oddi or otherwise destroy its function. But, in any measures, the dangers of (1) open the duodenum to cut the sphincter, (2)the difficulty of cutting the sphincter with certainty transduodenally and (3) cholangitis from duodenal reflux. (10)

In this article, the author suggested to treat the cases in which a possible common passage way between the biliary and pancreatic ducts especially for those who had previous operations, as removal of gall stones, cholecystostomy or choledochostomy with drainage of biliary tract for a long period, cholecystectomy or cholecystogastrostomy or in cases with normal biliary tract with endocholedochal sphincterotomy in order to prevent the recurrent attacks and the severe complications of recurrence of acute pancreatitis as indigestion, diabetis and pancreatic cyst, and the lethal outcome. In one of his reported cases, a woman with history of a cholecystectomy 20 years previously for attacks of severe epigastric pain with the recurrent attacks of severe epigastric pain and jaundice. He did try such operation, during that operation, the remnant of the cystic duct was opened and a catheter tied into it. Lipiodal was injected slowly into the biliary tract, simultaneously 5 c.c. of 1/10 N HCl was

injected into the duodenum by means of a duodenal tube passed preoperatively. This was done to produce spasm of the sphincter of Oddi without producing contraction of the duodenal musculature, in order to set up the optimum condition for visualization of the pancreatic duct. A cholangiogram obtained under these conditions clearly showed the pancreatic duc and proved the presence of reflux. The sphincterotome was passed into the duodenum through the common duct and the sphincter sectioned. A T-tube was placed into the common bile duct and the abdomen closed. 2 weeks later a duodenal tube was passed and cholangiographic studies were performed again, lipiodol could be seen to enter the duodenum easily. When HCl was injected into the duodenum by means of a duodenal tube, spasm of the muscularis mucosa of the duodenum occurred, but the pancreatic duct could not be visualized. These findings clearly show that under the same conditions, reflux of lipiodol into the pancreas did not occur. Close observation of the cholangiogram also showed that the intra-mural part of the common bile duct was intact. Spasm of the duodenal wall produced by injection of morphine, completely closed this intra-mural portion of the duct. Thus, destruction of the sphincter of Oddi, to prevent reflux of bile into the pancreatic duct, had been achieved without interferring with the mechanism which prevented duodenal reflux. It was evident that every time this patient's duodenum contracted the intra-murel part of the bile duct was compressed and acted as a one-way valve. For this operation, a perfect instrument has been designed by Colp, which would section the sphincter of Oddi safely through opening in the common bile duct and not through the duodenum, and at the same time would not cut the duodenal musculature. Therefore, he concluded that:

(1) Reflux of bile into the pancreatic duct should be prevented by cutting of the sphincter of Oddi.

(2) Endocholedochal sphincterotomy can be performed with safety without danger of producing cholangitis by duodenal reflux.

(3) Endocholedochal sphincterotomy should be the definitive surgical procedure in the treatment of recurrent acute pancreatitis if reflux of bile into the pancreas through a common biliary pancreatic passage way can be demonstrated.

Fundamentally, the function of the sphincter of Oddi is to increase the resistance to the flow of bile into the duodenum to an extent just sufficient to fill the gall bladder, therefore destruction of the sphincter leads to complete loss of function of gall bladder. It ceases to fill and therefore to empty. On physiological principles, when the sphincter of Oddi is destroyed, the gall bladder should be removed. This point should be considered in any future work on sphincter of Oddi. (10)

CASE ANALYSIS

Table I Age incidence

BB

Age	Number of cases	%
16-20yrs.	3	25
21-25	2	16.6
26-40	1	8.3
41-45	2	16.6
46-50	2	16.6
51-60	2	16.6

Table II. Sex Incidence

Sex	Number of cases	%
Male	7.	58.4
Female	5.	41.6

Table III. Symptomatology

Symptoms	Nature		No. of cases	%
Pain	Onset	Sudden	11	91.7
		Gradual	1	8.3
	Duration	Within 36 hrs.	6	50.0
		More than 36 hrs.	6	50.0
	Location	Epigastrium	9	75.0
		Other places	3	25.0
	Severity	Severe	11	91.7
		Mild	1	8.3
	Character	Parosysmal	4	33.4
		Continuous	4	33.4
		Colicky	6	50.0
		Other types	4	33.4
Anorexia, Nausea and Vomiting	Present in		12	100.0
Chill & fever	Present		4	33.4
Chilliness only	present		2	16.6
Feverishness only	present		1	8.3
Bowel movement	Constipation		8	66.6
	Loose stool		3	25.0
	Normal		1	8.1

— 23 —

— 24 —

Table IV. Physical signs.

Signs			No. of cases	%
Mentality	Clear		10	83.4
	Dull		2	16.6
Cyanosis			1	8.3
Juandice			1	8.3
Contour of the abdomen	Distended		5	41.7
	Full		3	25.0
	Soft		4	33.4
Rigidity	Present		9	75.0
	Location	Epigastrium	4	33.4
		Upper abdomen	3	25.0
		Whole abdomen	4	33.4
	Severity	Severe	2	16.6
		Marked	3	25.0
		Mild	4	33.4
Tenderness	Present		12	100.0
	Location	Epigastrium	6	50.0
		Upper abdomen	6	50.0
		Mild abdomen	2	16.6
		Whole abdomen	3	25.0
	Severity	Severe	5	41.6
		Marked	3	25.0
		Mild	4	33.4
Rebound tenderness	Present		4	33.4
	Location	Epigastrium	2	16.6
		Upper abdomen	2	16.6
		Mild abdomen	1	8.3
		Whole abdomen	0	0.0
	Severity	Severe	1	8.3
		Marked	2	16.6
		Mild	1	8.3
Gargling sound	Active		2	16.6
	Deminished		5	41.6

Table V. Temperature, Pulse and Respiration Chart.

		No. of cases	%
Temperature	Below 100.F.	6	50
	Below 102.F.	5	41.6
	Below 105.F.	1.	8.3
Pulse rate	Below 50/min.	3	25.0
	Between 70-100/min.	8	66.6
	Below 150/min.	1	8.3
Respiration	Below 25/min	10	83.4
	Above 25/min.	2	16.6

Table VI. Past History

Diseases		No. of cases	%
Similar attack	Present	6	50.0
	with jaundice	1	8.3
	without jaundice	11	91.7
Typhoid fever		2	16.6
Dysentery		2	16.6
Tonsillectomy		1	8.3

Table VII. Accompanied Diseases.

Diseases	Number of cases	%
Intestinal ascariasis	11	91.7
Cholecystitis	4	33.4
Cholangitis	2	16.6
Cholelethiasis	2	16.6
Latent syphilis	3	25.0

Table VIII. Laboratory Findings

Findings	Variations	No. of cases	%
W.B.C.	5,000-10,000	1	8.3
	10,000-15,000	4	33.4
	16,000-23,000	7	58.3
Polys	70-80%	3	25.0
	80-90%	4	33.4
	90-99%	4	33.4
Lymphocyte	less than 10%	6	50.0
	less than 20%	4	33.4
	less than 25%	1	8.3
Glucosria		1(trace)	8.3
Albuminuria	from + to +++	5	41.7
Blood amylase	has been done	4	33.4
	has not been done	7	58.3
	50-100 U.	1	25
	101 -300 U.	3	75.0
Urine amylase	has been done	10	83.4
	has not been done	2	16.6
	less than 1,0000.	8	80.0
	more than 1,000 U.	1	10.0
	more than 2,500 U.	1	10.0

Table IX. Treatment

Treatment	No. of cases	mortality	%
Surgical	8.	3.	37.5
Conservative	4	0	

Table X. Operations

Operation	Number of cases	Mortality
Cholecystectomy	5	None
Choledochostomy	2	None
Exploration and biopsy	2	2
Incision and drainage	1	1

Table XI. Operative findings

Findings	No. of cases	%
Fluid in the peritoneal cavity	2	25.0
Enlargemen and induration of pancreas	6	75.0
Fatty necrosis	3	37.5
Distended gall bladder	4	50.0
Dilated common bile duct	2	25.0
Contracted gall bladder	1	12.5
Gall stone	2	25.0
Abscess	1	25.0

Table XII. Post-operative complications

Complications	Number of cases
Acute dilatation of stomach	1
Lobar pneumonia	1
Bronchopneumonia	2

Table XIII. Result of treatment

Treatment	No. of cases	Complete cure	Death	Recurrence	Cause of death
Conservative	3	3	0	0	
Surgical	8	5	3	1	Toxemia Respiratory failure

Discussion

Illingworth and Dick said that acute pancreatitis affects both sexes equally and most frequent between the age of fifty and sixty. According to Popper's series, the sex incidence was definitely predominate in female while from the given 12 cases, 7 of them are male and the age incidence is about equally common in the age between 16 and 25 years and the age from 41 to 60 years.

From the cases analysis here, the onset was sudden in more than 90% of cases and half of them came within 36 hours after the onset, 91.7% of these cases experienced severe abdominal pain and in 75% cases, the pain was localized in the epigastrium and half of them were colicy in nature. Anorexia, nausea and vomiting are 100%, while there are only 7 cases had some chilly and feverish sensation, more than 66% of cases had constipation. Physically, 75% cases showed definite rigidity over the upper abdomen, usually marked in the epigastric region but are not severe in majority of cases. All of them showed tenderness in the upper abdomen and severe or marked ones in 66% [redacted]. The rebound tenderness is not very common, only present in 33.4% of cases, 8 patients had definite fulness of abdomen and only 5 of them showed marked distension. Gurgling sound deminished in 51% of these patients. Cyanosis had been recognized only in one of these cases. The mentality is clear in 10 of them. Most of these cases with slightly elevated temperature (below 102 F) normal pulse rate and normal respiration rate.

Regarding the past history, 6 patients had history of similar attack [redacted] or had painful attacks over the right upper abdomen which strongly suggested to be some kind of biliary system trouble, but, in detail, there was only one gave definite history of jaundice, even though, in a few cases, the icteric index detected during the hospitalization days was definitely higher than the so-called latent icterus level. From such picture, I agree with Popper's opium: " A history of previous gallbladder attacks or epigastric pain can be decided in more than half of the cases and the patients are sub-icteric, but as a rule have no distinct jaundice". Clinically as well as the operative finding, and the pathological reports are further proving that these cases had some kind of biliary tract trouble as cholecystitis, cholangitis, etc. but only two of them had cholelithiasis. Besides that, there are 11 cases accompanied with intestinal ascariasis.

From the laboratory findings, while blood cell count varies from range to moderate leukocytosis but more than half (7 cases) showed a white count varing from 16000 to 23000 per c.m.m. and all of them are predominated by polys. According to A.F. Herfort an absolute lymphopenic had been found in all 29 cases he studied while in these 12 cases, the lymphocyte count is less than 20%. Urine routine examination, revealled glucosuria present only in one case while 5 cases had albuminuria.

Blood amylase test had only been done in 4 cases. 3 of them are more than 100 U. while the rest one is only 64 units. Urine amylase increased in 80% of cases, varing from 128 to 640 units, the highest one is 2560 units, the lowest one is only 32 units, averagely, about one week after the first examination, urine amylase had been checked again in 4 of these cases and 3 of them showed very marked reduction. As we know, the elevation of the amylase level both in the blood and in the urine means that there is over absorption of amylase from the pancreas into the circulatory system resulting either from obstruction of the natural pancreatic drainage way or from over production resulting from some chemical, mechanical or biological irritation. So far as we know, the elevation of amylase in the blood or the urine, in the present time, is one of the diagnostic point for acute pancreatitis. According to Winslows method, as we used here, the normal blood amylase level ranges from 8 to 64 units, while the normal urine amylase level varies from 8 to 32 units.

All the 12 cases were diagnosed upon the history clinical manifestations and laboratory finding and the diagnosis were further more proved by the operative findings as free fluid in the peritoneal cavity, necrotic spots of the omentum and enlargement and induration of the pancreas itself etc. and the pathology of the pancreas.

In the 12 cases, 4 of them were treated conservatively with intravenous hydration, sedatives and analgesics as morphine and antispastic drugs as atropine and chemotherapy who gave excellent result and no one died from this treatment, in that group one patient was discharged less than 20 hours after admission because of against of operation. All the rest patients were intervened with surgery but only one had been operated with incision and drainage of the pancreas directly because of abscess formation, 5 of them were operated with cholecystectomy who showed excellent result also, gall stones were found in 2 of those 5 cases, and in such cases choledochostomy were done simple or multiple cigarette drains were applied in these 5 cases but 3 of them the foramen Winslow was drained. The rest 2 cases, who were treated with exploratory laparatomy and biopsy, died. one who came in 6 days after the onset, died of severe toxemia and respiratory failure 4 hours after the operation. Probably the most reasonable cause for the death of that patient was the poisonous split-protein products formed as the result of partial digestion of the pancreatic tissue and the products were rapidly absorbed and that together with the profound degree of shock. The other also died of toxemia and lobar pneumonia which developed 36 hours after operation. The one who treated with incision and drainage died of same reason but 6 days after operation.

The post-operative complications in the given cases are chiefly pneumonias, one developed bronchopneumonia and finally recovered while the another one developed lobar pneumonia and finally died of it, one complicated with suppuration and abscess formation. There was no one complicated with fistula formation, as Mc Cangham and Purcell mentioned that fistula formation after the operation of the pancreas was very common, but these 12 given cases just showed an absolutely reverse result, that may be due to the indirect drainage of the pancreas.

There is only one surgical case developed twice of recurrent similar attack with in a few months after cholecystectomy which were curred by conservative measures.

The mortality rate of both the surgical and conservative cases as a whole is 25%. There was no one died from conservative treatment while the mortality rate of the surgical cases alone is 37.5%.

In the so-called sulfa-drug and penicillin stage, to me the treatment of the future cases of acute pancreatitis will be much better conservatively rather than surgical intervension for the acute single cases, but in patients who complicated with biliary tract trouble, early operation is indicated. In the emergency cases, of course, the surgical intervension is the first choice but anyhow we should do a urgent blood or urine amylase test as well as the energency fluoroscopy in order to rule out other energency posibilities as perforation of peptic ulcer, intestinal obstruction etc. If we can rule out the other posibilities rather than acute pancreatitis and not complicated with other complications as biliary tract trouble. I would like to try conservative treatment first and watch the real indication very closely as failure of the conservative treatment.

Conclussion

12 cases of acute pancreatitis were collected and studied and revealed this diseases affects both male and female about equally, and commonly occurred among the age between 16-25 and between 41-55 years. Onset is sudden but shock and board like rigidity of abdomen were not sofpequent as some others mentioned Impairment of appetite, nausea and vomiting and constipation are the rule. Chills and fever frequently occur. Half of the cases had history of suggestive ~~of~~ biliary trouble but Jaundice rarely present. Moderate leukocytosis predominent by polys and relatively lymphopenia occurred in more than 90% of cases. Elevation of blood and urine amylase are characteristic. Glucouria is rare while albuminuria is present in about 41% of cases. 4 of them were treated conservatively with excellent results. 8 cases were treated surgically, operations were chiefly upon the biliary system w/ cholecystectomy, choledochostomy etc. The operative findings and the pathological reports proved the diagnosis. History of biliary tract trouble occurred in more than half of these cases but gall stones had only been demonstrated in 2 cases. The only one complication is pneumonia. Mortality of the cases as a whole was 25% while it was about 37.5% among the surgical cases. The cause of death were toxemia and respiratory failure. Recurrence had been occurred in one patient for 2 times within 2 months after operation. For proved acute cases with history of similar attacks before or history of biliary disturbance, it is more, to me, in favor to try conservative measures, otherwise, we should do surgical intervension as early as possible.

Reference

1. Text-book of Anatomy Cunningham 7th edition 1937 62, 642, 645, 665, 1411, 1462.
2. Text-book of Anatomy Gray's 28th edition 1945 1363-1367
3. Text-book of Histology Bremer & Weatherford 6th edition 1944 388-395
4. Applied Physiology Samson & Wright 7th edition 1945 613-617
5. Text-book of Pathology Boyd 3rd edition 608-609
6. Text-book of Surgical Pathology Ellingworth & Dink 5th edition 1945 581-585
7. Text-book of Surgery Homan's 6th edition 1945 892
8. Text-book of Surgery Christopher 3rd edition 1944 1337
9. Diseases of gall bladder and bile ducts Walters & Snell 1940 132, 329, 336.
10. S.G.O. Mar. 1948 Vol. 86 No:3 P. 295.
" The surgical treatment of recurrent acute pancreatitis by endocholedochal sphincterotomy" Henry Doublet & John H. Mulholland.
11. S.G.O. Dec. 1947 Vol. 85 No: 6 P. 794 "Acute Pancreatitis" with "An experimental study with special reference to X-ray therapy" Tegne C. Chisholm.
12. The American Journal of Digestive Diseases Jan. 1948 Vol. 15 No:1 P 1-4 "Acute Pancreatitis" H.L. Popper
13. The American Journal of Digestive Diseases Jan. 1948 Vol. 15 No:1 P 5-6 "Contribution to the Diagnosis of Acute Pancreatic Necrosis". K.F. Herfort.
14. The American Journal of Digestive Diseases Jan. 1947 Vol. 14 No:1 P 24-26 "Diagnosis of the diseases of the pancreas". Rudolf Schmann.
15. Archives of Surgery May 1941 Vol. 42 No:5 P 821-823 "Surgical lesion of the pancreas" Walters & Cleveland.
16. Archives of Surgery Aug. 1941 Vol. 43 No:2 P. 278-279 "Pancreatic Fistula" Mc. Cangham and Purcell.
17. S.G.O. Dec. 1947 Vol. 85 No:6 P.742-746 "The treatment of pancreatic pain by splanchnic nerve section." Geza De Takats.
18. S.G.O. April 1945 Vol. 80 No:4 P. 435-440 "An evaluation of the clinical significance of serum amylase and lipase determinations." Mc Call.
19. Lewis Practice of Surgery 1945 Vol VII P 1 & 43
20. Nelson's Surgery 1928 Vol. V P. 424.